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Biokinetics of Lead during Pregnancy

KATHRYN R. MAHAFFEY

National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina 27709

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Lead is readily transferred from the mother to the developing infant during pregnancy. The cord blood lead concentration is approximately 85–90% as high as the mother's blood lead concentration. Maternal blood lead declines slightly during the course of pregnancy; somewhat in excess of the decrease (Alexander and Delves, 1981) due to physiological hemodilution of pregnancy. Lead accumulates in the placenta and fetus. Occasionally the placental lead concentrations will be disproportionately higher than expected based on either maternal or fetal lead concentration. This may reflect a dysfunctional placenta (Korpela *et al.*, 1986).

The greatest elevation in lead concentrations has been reported in growth-retarded fetuses (Ward *et al.*, 1987). Whether or not this represents lead-induced growth retardation or accumulation of lead in the placenta of the fetus under stress is not clearly established. The latter interpretation is less likely because increased concentrations of calcium in the placenta of growth-retarded fetuses were not observed.

Data on lead concentrations in human fetal tissues are extremely limited. However, the pattern of lead transfer to the fetus closely mirrors the mineralization of fetal tissue (Horiuchi, 1965; Barltrop, 1974; Casey and Robinson, 1978) with lead being concentrated in the bone. Slightly later in gestation, lead is detected in other tissues including the brain, liver, and heart. Data demonstrating the presence of lead in fetal tissues reflect the quantitation limits of the analytical methods available at the time these data were gathered. There is no reason to believe lead does not occur in all fetal tissues from conception onward.

Maternal bone can act as a source of mineral for the developing fetus. The main stimulus for mobilization of maternal bone mineral is maintenance of a stable concentration of serum-ionized calcium. Metabolic studies have evaluated the changes in maternal calcium biokinetics associated with pregnancy. During pregnancy there is an increase in the miscible calcium pool at term and an approximate doubling of calcium pool turnover and bone mineral accretion rate. Calcium absorption is increased from the earliest period of pregnancy and remains high throughout gestation (Henry and Skillman, 1971). Bone turnover is increased during pregnancy; whether or not there is skeletal loss of mineral depends upon the calcium content of the diet (Henry and Skillman, 1971).

Most research on transfer of lead to the fetus has focused on lead exposure concurrent with or immediately prior to the pregnancy. However, it is important to know the extent to which maternal body burden of lead, incorporated in the years prior to pregnancy (e.g., from childhood lead poisoning), is bioavailable to the fetus. Data from experimental animals

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(Buchet *et al.*, 1977; Keller and Doherty, 1980) indicate that lead is mobilized during pregnancy from body stores incorporated prior to pregnancy. The clinical literature contains case reports of this phenomena in humans (Thompson *et al.*, 1985). The proportionate contribution of maternal lead stored prior to pregnancy to the fetal body lead burden is an important tissue for ongoing research to address.

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